

CORRELATION BETWEEN PERCUTANEOUS TRANSHEPATIC PORTOGRAPHY
AND CLINICAL FINDINGS IN 56 PATIENTS WITH
PORTAL HYPERTENSION

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ABSTRACT

56 consecutive patients with portal hypertension were studied with percutaneous transhepatic portography and the results were correlated to clinical findings and the number of upper gastrointestinal haemorrhages and the size of the individual bleeding.

An abundance of collateral paths was noted in most patients. No regularity in development of these collaterals was found. It was not correlated to liver disease etiology, sex or liver function parameters. Portal pressure was not correlated to the size or amount of collaterals.

In four patients with liver cirrhosis hepatofugal flow in one segment of the liver was noted proving that portal flow through the liver is not uniform in this disease.

The size of the haemorrhages was only correlated to presence of hepatofugal flow in the main stem of the portal vein. It was not correlated to the estimated size of the oesophageal varices or to portal pressure.

Percutaneous transhepatic portography seems to be of little help in selecting "high risk bleeders" in portal hypertension.

Other factors may be of greater help in this task as indicated by the findings in this investigation that patients with alcohol cirrhosis had larger haemorrhages than those with cirrhosis of other etiology and that patients with none or few bleeding episodes had higher thrombocyte count than those with several haemorrhages.

The principles of managing gastrointestinal haemorrhages in patients with portal hypertension is becoming increasingly unclear since creation of a portacaval shunt in every case is questionable and there is a growing dissatisfaction with the longterm results of this operation. Controlled clinical trials has not been able to show increased survival following shunt operation neither prophylactically (Conn et al 1972; Resnick et al 1969) nor therapeutically (Resnick et al 1974; Jackson et al 1973; Rueff et al 1974).

Many patients with known cirrhosis of the liver succumb in their first gastrointestinal bleeding. To identify these high risk patients and prevent their haemorrhage is important but still not possible due to our defective knowledge of what makes a patient with portal hypertension bleed.

In Europe revival of the old method of transoesophageal injection of sclerosants to prevent further bleeding is obvious, (Johnston & Rodgers 1973; Paquet & Engel 1974). A new method for control of haemorrhage from oesophageal varices via transhepatic injections into the coronary vein has also been developed (Lunderquist & Vang 1974).

Thus it may be possible that selection of patients to various therapy is becoming more and more important but our tools for this selection are very imprecise. To develop them must be an important task in management of patients with portal hypertension.

A relatively recent development of the technique to study the portal vein and its branches is percutaneous trans-hepatic portography. It was first described by Bierman (1952) and technically refined by Wiechel (1971). This method offers a unique possibility of placing catheters in different parts of the portal system and thus obtaining detail studies of separate parts of this system. This makes a much better visualization of the different portosystemic communications possible.

The present investigation is an attempt to find out:

1. What collateral patterns could be seen with this new technique?
2. Do these patterns correlate in any way to important clinical findings?
3. Can it be used as a tool for selecting "high risk" patients in portal hypertension?

MATERIAL AND METHODS

The 56 patients included in this investigation are the first 56 patients with portal hypertension investigated with percutaneous transhepatic portography (P.T.P.) at our hospital. Only cases, where no specific therapy against the portal hypertension, such as surgical shunts or sclerosing injections, had been instituted, were included. Generally the indication for the investigation was upper gastrointestinal bleeding with suspected or known portal hypertension. In some patients more than one investigation was done, however, only the first was included in this study. Some patients were investigated because of suspected liver tumors and found to have portal hypertension and cirrhosis.

In many cases the investigation was combined with sclerotherapy against bleeding varices through the coronary vein. The result of this is described in another paper (Lunderquist et al 1977).

37 of the patients were men, 19 were women. Mean age was 56.9 ($\pm$ 11.9). The women were significantly older than the men, mean values 66.7 ($\pm$ 12.8) years versus 53.4 ($\pm$ 10.4) years ($p < 0.05$ ANOVA).

35 patients were classified as alcoholics (29 men and 6 women), 6 were classified as having cirrhosis due to chronic aggressive hepatitis (all women), 1 man had a secondary biliary cirrhosis, 13 patients had a cryptogenic cirrhosis (6 men and 7 women) and 1 man had a primary portal hypertension with repeated normal liver biopsies and normal laparoscopic picture and no signs of portal vein occlusion at the X-ray investigation.

31 of the patients had a known liver disease duration of less than one year, 16 from 1 to 5 years and 8 more than 5 years. This was not correlated to liver disease etiology or age of the patient. Our P.T.P. technique has recently been extensively described (Hoevels et al 1977) and therefore no description is included here.

In the interpretation of the portograms and records the following points were evaluated:

1. Estimated flow through oesophageal varices arbitrarily classified as 0, 1, 2, 3.
2. Presence and size of coronary vein measured in millimeters half a centimeter from its connection with the portal vein.

3. Filling of the oesophageal varices from the coronary vein only, short gastric veins only, both coronary vein and short gastric veins.
 4. Portal pressure measured with an open manometer. As zero-point was used the level of the portal vein in the liver hilus.
 5. Direction of flow in the main stem of the portal vein.
 6. The diameter of the portal vein at its beginning and of the splenic vein at its junction with the superior mesenteric vein.
 7. The presence of retrograde flow and largest diameter of the inferior mesenteric vein and the umbilical vein.
 8. Visualization of posterior superior pancreaticoduodenal vein (P S P D), upper and lower splenic hilus collaterals, a retroperitoneal plexus, the left renal vein and intercostal veins.
 9. Segmental intrahepatic hepatofugal flow.
- Based on the registration of collateral paths the patients were classified into the following categories in a variable called "collateral pattern".
- a. Having the coronary-oesophageal path only.
 - b. Having the coronary-oesophageal path + one other main collateral path.
 - c. Having the coronary-oesophageal path + two other main paths.
 - d. Having the coronary-oesophageal path + three or more other main paths.
 - e. Not having the coronary-oesophageal path.

Furthermore the patients were separated into two classes according to the presence or absence of at least one really large collateral outside the oesophageal-gastric area defined as

- a. An umbilical vein more than 8 mm in diameter.
- b. Retrograde flow in an inferior mesenteric vein of more than 8 mm diameter.
- c. A spontaneous spleno-renal shunt more than 6 mm in diameter. These limits were chosen to classify about 1/4 of above mentioned collaterals as "large".

To evaluate the patients individual risk of having an upper gastrointestinal bleeding their records were examined and their separate bleeding episodes up to the date of the P.T.P. counted. Sometimes it was difficult to decide if an individual episode was single or in fact was a re-bleeding. An arbitrarily limit of 48 hours freedom from bleeding was set. If a re-bleeding occurred within 48 hours the patients were considered to have had one bleeding episode. To get an idea of how big each separate bleeding episode was we counted the units of blood transfusions given in the last bleeding episode prior to the

P.T.P. These two numeric informations (number of gastro-intestinal haemorrhages + number of transfusions) were also combined to a classification scale with the following steps:

1. No upper gastro-intestinal bleeding.
2. One minor bleeding.
3. More than one minor bleeding.
4. One life-threatening bleeding.
5. More than one life-threatening bleeding.

The limit between minor and life-threatening bleeding were chosen as transfusion of less than four units of blood the first 24 hours or altogether less than 6 units.

The records of the patients were also examined and laboratory data from the last bleeding episode indicating liver function and bleeding tendency were noted (albumin/s, bilirubin/s, thrombocyte count, APTT (activated partial thromboplastin time), NT (Normotest, Nyegaard & Co A/S, Oslo, Norway). Only values recorded within the first 24 hours after the bleeding started were accepted.

Also "elective" values of the above mentioned parameters were recorded. As "elective" were considered the first values taken after admittance to the hospital with no clinically overt bleeding. This admittance was as close as possible to the P.T.P. examination but not longer before or after the investigation than two months.

Noted in the records were also the degree of encephalopathy classified into three classes:

- a. None b. Possible c. Definite

The encephalopathy were recorded in three different ways:

- a. Clinical b. EEG c. Psychometric tests.

In all these ways the three above mentioned classes were used. Regarding EEG and psychometric tests the report from the neurophysiologist respectively the psychologist were used. In the clinical classification possible encephalopathy were cases with noted slight sensory impairment of doubtful etiology or patients that otherwise were very difficult to evaluate due to for instance combined alcoholic hallucinations. Also taken from the records were data regarding sex, age, duration and etiology of the liver disease, presence of ascites at the investigation.

Based on the above mentioned liver function parameters the patients were classified into liver function group according to the method outlined in table 1 (modified from J. Hobbs). The slight impropriety that one missing value for a patient were substituted with the mean of the four others were accepted to classify as many patients as possible. Patients with two missing values were not classified. As we had two

different sets of laboratory values for each patient, one acute and one elective, two different classifications were made, one elective and one acute.

Statistical evaluation: The calculations were made with help of a Hewlett-Packard calculator (HP-9830). Varying statistical programs all separately constructed for this investigation were then applied. To compare data on a nominal scale χ^2 -test or Fisher's exact test were used. If possible these tests were replaced by tests with stronger power by having the data on an interval or rank scale. Adaption to the normal distribution was tested with the Kolmogorov-Smirnoff test and the probit-paper-test. To test correlations in data where both parameters were measured on an interval scale an ordinary linear regression were used. In one of the parameters were on an interval scale and the other on a normal scale one-way analysis of variance (ANOVA) were used. If data from both parameters were on a rank scale Spearman's rank sum test were used. If one of the data set were in a rank scale and the other on a nominal scale Kruskal-Wallis test were used. In all these tests $p = 0.05$ was accepted as the highest significance level.

RESULTS

Clinical findings

27 patients had ascites at the time of the investigation, in another 6 ascites had been noted earlier in their history and in 22 there were no history of ascites. No correlations were found between this parameter and age, sex or etiology of the liver disease.

Encephalopathy was noted clinically with certainty in 13 patients, probably in 4 and not in 38. In EEG it was noted with certainty in 11, probably in 7 and not in 15 and with psychometric tests with certainty in 9, probably in 5 and not in 10 patients. This was not significantly correlated to age or sex of the patient or liver disease etiology.

Liver function: Based on elective liver values 11 patients were classified in group A, 17 in group B and 11 in group C. Based on acute liver values 6 patients were classified in group A, 6 in group B and 20 in group C.

Liver and coagulation laboratory parameters were also calculated as indicated above but the results are omitted here.

Haemorrhages: 14 of the patients had no upper gastrointestinal bleeding, 42 had from 1 to 12 episodes. Median value of all patients were 2 with lower quartile 1 and upper 3.

Transfusions: Median value of number of units of blood transfused at the last bleeding episode prior to the X-ray investigation were 6 (lower quartile 4, upper quartile 11).

Classification of bleeders: 14 had no haemorrhage, 3 had one minor bleeding episode, 7 had more than one minor episode, 10 had one large episode and 21 more than one life-threatening episode.

Portography findings

Portal pressure: Mean value $\pm$ SD = 33.8 ± 6.7 cm H₂O. Range 22 - 56 cm H₂O.

Direction of flow in the portal vein: Hepatofugal flow was noted in 7/54 cases.

Size of oesophageal varices: No varices could be seen in 9 cases. The varices were classified as small in 19 cases, of medium size in 17 cases and large in 9.

The coronary vein: Served as a collateral in 46 cases. The median diameter of the vein was 8 mm. (Upper quartile 10 mm, lower 6 mm.)

The inferior mesenteric vein was not visualized in 17 cases. It acted as an outflow tract in 37 cases. Median maximal diameter was 8 mm. (Lower quartile 7 mm, upper quartile 9 mm.)

The posterior superior pancreatico duodenal vein (PSPD) acted as an outflow tract in 13 cases. It was not visualized in 41.

Upper splenic hilus collaterals were seen in 7 cases, not visualized in 47.

Lower splenic hilus collaterals were seen in 14 cases, absent in 40.

The umbilical vein was seen in 16 cases and absent in 38. The median maximal width was 7 mm (lower quartile 5 mm, upper 12 mm).

Segmental hepatofugal flow in the liver were seen in 4 cases, (see figure I). This phenomena was not correlated to any specific flow pattern.



Fig. I. 59-year-old woman with liver cirrhosis and portal hypertension. Transhepatic portography. Segmental intrahepatic hepatofugal flow (arrow) demonstrated by none-filling of branch to dorsocaudal segment.

The oesophageal varices were in 17 cases filled only via the coronary vein, in 4 cases only via the short gastric veins. In 23 cases both systems contributed to the filling of the varices.

A retroperitoneal plexus could be seen in 21 cases but not in 33.

The left renal vein were filled in 20 cases and could not be seen in 34.

Intercostal veins were seen filled in 16 cases and not seen in 38.

The collateral pattern classification showed that 7 patients had coronary collaterals only, 18 had coronary + one major collateral pathway, 9 had coronary + two other pathways, 11 coronary + three or more main collateral pathways. 9 had no coronary collaterals.

Large collaterals: 17 were classified as having large collaterals, 37 as not having them.

Relations of portal pressure to bleeding parameters and portographic flow patterns

No relation could be seen between portal pressure and different bleeding parameters. Patients with massive bleedings had the same portal pressure as those with minor or no bleeding at all. Likewise no correlation was found between portal pressure and any specific portographic picture. No differences in portal pressure were seen between the groups with large or small varices, large or small coronary vein, large or small general collaterals or extensive or small collateralization. There was a significant pressure difference between patients with hepatofugal flow in the portal vein and those with hepatopetal flow but the significance was only marginal ($0.01 < p < 0.05$), and the difference between group means (38 ± 6 cm H₂O versus 33 ± 6 cm H₂O) was small.

Portal pressure was not significantly correlated to liver function values although a weak tendency was noted that bilirubin was higher and albumin lower in patients with high portal pressure.

Relation of collateral patterns to haemorrhages

The size of the varices or the size of the coronary vein was not related to bleeding parameters. Nor was there any

relation between the amount of bleeding and if the blood to the varices came from the coronary vein or short gastric veins or both.

Presence or absence of large collaterals, or having many or few collateral systems were not correlated to any bleeding parameter. However, hepatofugal flow was related to a large number of transfusions during the last bleeding before the X-ray investigation ($p < 0.01$ Fisher's exact test). No statistically significant correlation to the number of haemorrhages were seen but 6/7 patients with hepatofugal had had haemorrhages compared to 34/47 in patients with hepatopetal flow.

Presence of the umbilical vein was associated with significantly fewer number of bleeding episodes ($0.01 < p < 0.05$ ANOVA). No other X-ray pattern could be seen to correlate with any of the bleeding parameters.

Relation between haemorrhages and other clinical findings

Bleeding tendency was furthermore only correlated to two parameters:

1. Alcoholics had larger haemorrhages than nonalcoholics as judged by a larger number of transfusions in the last recorded bleeding episode. 16/22 had 6 or more units of blood compared to 4/13 ($p < 0.05$ Fisher's exact test).

2. Number of bleeding episodes were correlated to the number of thrombocytes (elective) ($p < 0.05$ Spearman's test). This correlation was mainly due to that patients with no haemorrhages had higher thrombocyte count than patients with one or more bleeding episodes.

Relation of different collateral patterns

The size of the oesophageal varices as judged by the porto-gram did in no way correlate to any other X-ray parameter, but to the size of the coronary vein. Very large varices were only seen when the coronary vein was more than 4 mm in diameter. This difference was statistically significant ($p < 0.001$ χ^2 -test). The size of the coronary vein was not correlated to the size of any other investigated vein or flow pattern.

No certain correlation between different flow patterns was established except that the visualization of a retro-peritoneal plexus was correlated to filling of one of its

important feeding veins PSPD ($p < 0.05$ χ^2 -test). For instance presence of an umbilical vein or inferior mesenteric vein neither increased or reduced the chance of also having a left renal vein visualized.

Relation between collateral pattern and clinical findings

No correlation between liver function and flow pattern could be seen but in many tests the groups were too small for evaluation. For instance all five patients with hepatofugal flow were classified in the worst liver function group according to the above mentioned criteria but as only five patients with this type of flow were evaluated in this comparison the difference from the patients with hepatopetal flow never reached statistical significance. However, even in the more "easily" evaluated parameter albumin/s could no difference between groups with hepatofugal and hepatopetal flow be found.

The higher acute bilirubin in the group with hepatofugal flow ($p < 0.05$ Kruskal-Wallis test) is probably explained by the larger haemorrhages in this group.

Presence of encephalopathy was statistically significantly correlated to hepatofugal flow both clinically ($p < 0.01$ χ^2 -test) and in EEG ($p < 0.05$ χ^2 -test). Psychometrical evaluation was not possible as only two patients with hepatofugal flow came to such a test. No other significant correlation to collateral pattern was seen.

The width of the portal and splenic vein was neither correlated to any specific flow pattern nor to any liver function test. However, a larger width of the splenic vein was seen in patients with lower number of thrombocytes ($p < 0.05$ Kruskal-Wallis test).

DISCUSSION

The introduction of methods for roentgen examination of the portal vein system as splenoportography (Boulvin et al 1951, Turner et al 1957), umbilical vein portography (Carbalhaes 1959, Bayly 1964) and percutaneous transhepatic portography (Bierman et al 1952) has greatly increased our knowledge of venous anatomy and pathophysiology in portal hypertension. The possibility of detail studies of the different parts of the portal venous system is probably greatest with the last mentioned method due to the relative ease



Fig. II. 53-year-old man with liver cirrhosis and portal hypertension. Transhepatic portography. a/ Early portographic phase. Retrograde flow through coronary vein and oesophageal varices, posterior superior pancreaticoduodenal vein and inferior mesenteric vein. b/ Late portographic phase showing large retroperitoneal plexus.

with that the injection catheter is manipulated to different part of the venous system (Viamonte et al 1975). The visualization of different parts of the portal system is, however, in this method as well as in the other X-ray techniques largely dependent on where in the portal vein system the injection of contrast medium is performed. Nonvisualization does not necessarily mean that the collateral path does not exist.

The most striking finding in the pathological anatomy of portal hypertension in this investigation was the abundance of collateral paths present in many patients. See figure II!

A spontaneous shunt to the left renal vein was visualized in 37% of the investigations, the umbilical vein acted as an outflow tract in 30%. The last is a much larger figure than known from spleno-portograms (Burchell et al 1970). A retroperitoneal plexus was seen in 39%. The coronary-oesophageal vein complex was seen in 85%.

The development of collaterals does not seem to be influenced by factors like sex, etiology of liver disease or liver function. No correlation between the amount of collaterals and these parameters was found. It is probable that the extent of collateralization depends more on the possibility to dilate the small already existing collaterals between the portal and caval system. The main collaterals in our investigation have been seen also in human beings without portal hypertension (Thamm 1940). The other factor that possibly influence the collateral development is liver resistance to portal blood flow which was not measured in our investigation. Ordinary liver function parameters like bilirubin/s obviously do not correlate so well with liver resistance that this could be seen in the correlations between collateralization and liver function.

Portal pressure bore no relation to the amount of collateralization or to the size of the coronary or oesophageal varices. Diverse meanings concerning this point has been expressed by other investigators. Lebrec et al (1976) found no correlation between portal pressure and degree of shunting using an isotope dilution technique.

Rousselot et al (1959) on the other hand found a correlation between portal pressure and degree of collateralization in splenoportograms, but this correlation is in their investigation undue strengthened by including subjects without collateralization and with normal pressure. The same objection seems to be guilty for the investigation of Viallet et al (1970) who found a very close correlation between portal pressure and size of coronary vein.

Our method of classifying the patients individual tendency to bleed is influenced by many factors not possible to evaluate in this type of investigation as for instance how quickly the diagnosis of the bleeding varices were made after the first haemorrhage, and different treatment of the individual haemorrhage. Due to this "noise" of un-evaluated variables a finding of a non-existent correlation should not be considered as a proof of that none exists. Acute endoscopy during active bleeding was not generally used in this investigation and was not evaluated. The difficulties in separating variceal from non-variceal bleeding in these patients can be considerable (Simert et al 1976) and in some materials only very few or none of the upper gastrointestinal haemorrhages really emanated from the varices (Borsari 1977). If this is the case also in our material probably very little correlation can be expected between the P.T.P. findings and the bleeding tendency of the patients.

Bearing those points in mind we still found a correlation between the finding of hepatofugal flow in the portal vein and the size of the haemorrhages. All patients with hepatofugal portal vein flow had large haemorrhages either due to direct influence on the bleeding rate or due to little response to the instituted therapy (vasopressin infusion as first step then Sengstaken balloon.

The association between presence of the umbilical vein and few or no gastrointestinal haemorrhages is probably a false association due to so called "mass significance" as all the correlation was seen in the group with the small umbilical vein and none in the group with a large vein.

There have been earlier trials to identify high risk patients with portal hypertension by roentgen investigations of the portal system. Jackson (1963) and Hassab (1967) reported that patients with "cephalic" collaterals in splenoportograms had a higher risk of bleeding than those with "caudal" collaterals. This conclusion is, however, poorly statistically supported and this pattern could not be seen in this investigation. It is possible that selection of high risk patients in portal hypertension is better done with other investigation as for instance coagulation factors (vide infra) or oesophagoscopy (Dagradi 1972).

"Large" collaterals could not in this investigation as well in that of Hamilton and Sullivan (1961) reduce the portal pressure or reduce the risk of gastrointestinal bleeding. However, to bear the flow of a portal vein two cm in diameter no less than 40 collaterals with a smallest diameter of 8 mm are needed according to Poiseuille's law. Very large haemodynamically active collaterals have unfrequently

been reported (Wexler et Mac Lean 1975), although none was seen in this investigation.

Another important finding was the fact that patients with alcohol abuse had larger haemorrhages than those with other cirrhosis etiology. A possible explanation to this may be the toxic effect of ethanol on coagulation mechanism. Alcohol is known to experimentally aggregate thrombocytes (Elmér et al, to be published) and alcoholics have reduced number of thrombocytes (Straus 1973). Alcohol has also probably an direct effect on the gastric mucosa that may induce larger haemorrhages.

In this study patients without or with only one haemorrhage had significantly higher thrombocyte count than those with repeated haemorrhages which also indicates the importance of coagulation factors in this connection.

The lack of correlation between portal pressure and haemorrhage size as well as other parameters could indicate the very restricted help this estimation is in patients with known portal hypertension. The degree of hypertension does not seem to be of any interest as far as we can see in this investigation. However, Jackson et al (1971) in their prospective study of therapeutic portacaval shunts could see an increased risk of re-bleeding in patients with high portal pressure in the medically treated group.

The phenomenon of segmental hepatofugal flow previously not reported as far as we know, but seen in four cases in this investigation proves that portal flow is not uniform through every part of the cirrhotic liver. Haemodynamic studies of portal flow based on catheters placed in a hepatic vein with an without injection of contrast medium must be regarded with utmost care due to this fact. However, wedge hepatic pressure readings have been proven to very closely correlate to pressure inside the portal vein (Viallet et al 1970).

Table 1.

Modified Child classification of patients with liver cirrhosis. Group A ≤ 6 points, group B 7 - 9 points, group C ≥ 10 points.

Points	1	2	3
Encephalopathy	0	slight	severe
Ascites	0	slight	severe
Bilirubin mg/100 ml	≤ 2.0	2.1-3.0	≥ 3.0
Serum albumin g/l	≥ 36	28-35	≤ 28
Normotest	≥ 70	40-69	≤ 40

REFERENCES

- Bayly, J.H. 1964. The use of the umbilical vein in the diagnosis of upper gastrointestinal bleeding. *Am. J. Gastroenterol.* 41; 235.
- Bierman, H.R., Steinbach, H.L., White, L.P., Kelly, K.H. 1952. Portal venipuncture. A percutaneous, transhepatic approach. *Proc. Soc. Exp. Biol. Med.* 79; 550.
- Borsari, G. 1976. Ösophagusvarizenruptur: Mythos oder Realität? *Z. Gastroenterol.* 14; 700.
- Boulvin, R., Chevalier, M., Gallus, P., Nagel, M. 1951. Contribution a l'étude du syndrome d'hypertension portale: étude clinique et portographique. *Acta Chir. Belg.* 50; 534.
- Burchell, A.R., Panke, W.F., Moreno, A.K., Rousselot, L.M. 1970. The patent umbilical vein in portal hypertension. *Surg. Gynecol. Obstet.* 130; 77.
- Carbalhaes, O.G. 1959. Portography, a preliminary report of a new technique via the umbilical vein. *Clin. Proc. Child. Hosp. (Wash.)* 15; 120.
- Conn, H.O., Lindenmuth, W.W., May, C.J., Ramsby, G.R. 1972. Prophylactic portacaval anastomosis - A tale of two studies. *Medicine* 51; 27.
- Dagradi, A.E. 1972. The natural history of esophageal varices in patients with alcoholic liver cirrhosis. An endoscopic and clinical study. *Am. J. Gastroenterol.* 57; 520.
- Elmér, O., Göransson, G., Saku, M., Bengmark, S. Ethanol-induced microaggregate formation in pig and rabbit blood. An in vitro study. To be published in *Thromb. Haemostas.*
- Hamilton, L.C., Sullivan, B.H. 1961. Natural portosystemic venous shunts in portal hypertension. *Med. Ann. District of Columbia* 30; 654.
- Hassab, M.A. 1967. Gastroesophageal decongestion and splenectomy in the treatment of esophageal varices in bilharzial cirrhosis: Further studies with a report on 355 operations. *Surgery* 61; 169.
- Hobbs, J. Personal communication.

Hoevels, J., Lunderquist, A., Tylén, U. Percutaneous transhepatic portography. Accepted for publication in Radiology.

Jackson, F.C. 1963. "Directional" flow patterns in portal hypertension. Arch. Surg. 87; 307.

Jackson, F.C., Perrin, E.B., Felix, W.R., Smith, A.G. 1971. A clinical investigation of the portacaval shunt. V. Survival analysis of the therapeutic operation. Ann. Surg. 174; 672.

Johnston, G.W., Rodgers, H.W. 1973. A review of 15 years experience in the use of sclerotherapy in the control of acute hemorrhage from oesophageal varices. Brit. J. Surg. 60; 797.

Lebrec, D., Kotelanski, B., Cohn, J.N. 1976. Splanchnic hemodynamics in cirrhotic patients with oesophageal varices and gastrointestinal bleeding. Gastroenterology 70; 1108.

Lunderquist, A., Vang, J. 1974. Transhepatic catheterization and obliteration of the coronary vein in patients with portal hypertension and esophageal varices. New Engl. J. Med. 291; 646.

Lunderquist, A., Simert, G., Tylén, U., Vang, J. 1977. Follow-up of patients with portal hypertension and esophageal varices treated with percutaneous obliteration of gastric coronary vein. Radiology 122; 59.

Paquet, K.-J., Engel, C. 1974. Die Wandsklerosierung des Ösophagusvarizenblutung und der drohenden Hamorrhagie bei dekompensierten Leberfunktion. Z. Gastroenterol. 12; 395.

Resnick, R.H., Chalmers, T.C., Ishihara, A.M., Garceau, A.J., Callow, A.D., Schimmel, E.M., O'hara, E.T. and the Boston Interhospital Liver Group. 1969. A controlled study of the prophylactic portacaval shunt. A final report. Ann. Intern. Med. 70; 675.

Resnick, R.H., Iber, F.L., Ishihara, A.M. and the Boston Interhospital Liver Group. 1974. A controlled study of the therapeutic portacaval shunt. Gastroenterology 67; 843.

Rueff, B., Prandi, D., Degos, F., Sicot, J., Degos, J.D., Sicot, C., Maillard, J.N., Fauvert, R., Benhamou, J.P. 1976. A controlled study of therapeutic portacaval shunt in alcoholic cirrhosis. Lancet 1; 655.

Simert, G., Bengmark, S., Börjesson, B., Eriksson, S., Hammar, E., Liedberg, G., Pettersson, K.-I., Vang, J. 1976. Oesophagit och cardiafunktion hos patienter med oesophagusvaricer. Svensk kirurgi 23; 146.

Straus, D.J. 1973. Hematologic aspects of alcoholism. Semin. Hematol. 10; 183.

Thamm, M. 1940. Die portacavalen Venenverbindungen des Menschen. Zentralbl. Chir. 39; 1828.

Turner, M.D., Sherlock, S., Steiner, R.E. 1957. Splenic venography and intrasplenic pressure measurement in the clinical investigation of the portal venous system. Am. J. Med. 23; 846.

Viallet, A., Legere, A., Lavoie, P.R. 1970. Hepatic and umbilicoportal catheterization in portal hypertension. Ann. NY. Acad. Sci. 170; 177.

Viamonte Jr, M., Le Page, J., Lunderquist, A., Peretros, R., Russel, E., Viamonte, M., Camacho, M. 1975. Selective catheterization of the portal vein and its tributaries. Preliminary report. Radiology 114; 457.

Wexler, M.J., Mac Lean, L.D. 1975. Massive spontaneous portal systemic shunting without varices. Arch. Surg. 110; 995.

Wiechel, K.L. 1971. Tekniken vid percutan transhepatisk portapunktion (PTP). Nord. Med. 86; 912.